

M. tuberculosis belonging to SIT 52 that caused tuberculous meningitis was reported in a human in Thailand (10), but that case was not related to the case reported here. Our finding of a relatively novel spoligotype of *M. tuberculosis* in an animal destined for the pet trade underscores the need for intensive testing of and extended quarantine for all imported nonhuman primates to prevent the spread of newly isolated *M. tuberculosis* (4,7,8).

Acknowledgments

We thank Areeya Disrattakit and Nampung Makao for their excellent technical assistance and Eric Lombardini and Roongroje Thanawongnuwech for their critical suggestions on this manuscript.

This report was financially supported by Grants for Development of New Faculty Staff, Ratchadaphiseksomphot Endowment Fund, Chulalongkorn University.

The authors declare no potential conflicts of interest with respect to the research, authorship, and/or publication of this article, and no competing financial interests exist.

References

1. Wilbur AK, Engel GA, Rompis A, Putra IG, Lee BP, Aggimarangsee N, et al. From the mouths of monkeys: detection of *Mycobacterium tuberculosis* complex DNA from buccal swabs of synanthropic macaques. *Am J Primatol*. 2012;74:676–86. <http://dx.doi.org/10.1002/ajp.22022>
2. Lin PL, Pawar S, Myers A, Pegu A, Fuhrman C, Reinhart TA, et al. Early events in *Mycobacterium tuberculosis* infection in cynomolgus macaques. *Infect Immun*. 2006;74:3790–803. <http://dx.doi.org/10.1128/IAI.00064-06>
3. Chaiprasert A, Prammananan T, Tingtoy N, Na-Ubol P, Srimuang S, Samerpitak K, et al. One-tube multiplex PCR method for rapid identification of *Mycobacterium tuberculosis*. *Southeast Asian J Trop Med Public Health*. 2006;37:494–502.
4. Panarella ML, Bimes RS. A naturally occurring outbreak of tuberculosis in a group of imported cynomolgus monkeys (*Macaca fascicularis*). *J Am Assoc Lab Anim Sci*. 2010;49:221–5.
5. Centers for Disease Control and Prevention. Tuberculosis in imported nonhuman primates—United States, June 1990–May 1993. *MMWR Morb Mortal Wkly Rep*. 1993;42:572–6.
6. Pavlin BI, Schloegel LM, Daszak P. Risk of importing zoonotic diseases through wildlife trade, United States. *Emerg Infect Dis*. 2009;15:1721–6. <http://dx.doi.org/10.3201/eid1511.090467>
7. Shipley ST, Coksaygan T, Johnson DK, McLeod CG Jr, DeTolla LJ. Diagnosis and prevention of dissemination of tuberculosis in a recently imported rhesus macaque (*Macaca mulatta*). *J Med Primatol*. 2008;37(Suppl 1):20–4. <http://dx.doi.org/10.1111/j.1600-0684.2007.00266.x>
8. Engel GA, Wilbur AK, Westmark A, Horn D, Johnson J, Jones-Engel L. Naturally acquired *Mycobacterium tuberculosis* complex in laboratory pig-tailed macaques. *Emerg Microb Infect*. 2012;1:e30. <http://dx.doi.org/10.1038/emi.2012.31>
9. Brudey K, Driscoll JR, Rigouts L, Prodinger WM, Gori A, Al-Hajj SA, et al. *Mycobacterium tuberculosis* complex genetic diversity: mining the fourth international spoligotyping database (SpolDB4) for classification, population genetics and epidemiology. *BMC Microbiol*. 2006;6:23. <http://dx.doi.org/10.1186/1471-2180-6-23>
10. Yorsangsukkamol J, Chaiprasert A, Prammananan T, Palittapongarnpim P, Limsoontarakul S, Prayoonwivat N. Molecular analysis of *Mycobacterium tuberculosis* from tuberculous meningitis patients in Thailand. *Tuberculosis (Edinb)*. 2009;89:304–9. <http://dx.doi.org/10.1016/j.tube.2009.05.001>

Address for correspondence: Sawang Kesdangsakonwut, Department of Pathology, Faculty of Veterinary Science, Chulalongkorn University, Henri Dunant Rd, Pathumwan, Bangkok 10330, Thailand; email: sawang.k@chula.ac.th

Treatment of *Mycobacterium abscessus* subsp. *massiliense* Tricuspid Valve Endocarditis

R. Gordon Huth, Elizabeth Douglass, Kristin Mondy, Sruthi Vasireddy, Richard J. Wallace Jr.

Author affiliations: The University of Texas at Austin Dell Medical School Residency Programs, Austin, Texas, USA (R.G. Huth, E. Douglass, K. Mondy); University of Texas Health Science Center, Tyler, Texas, USA (S. Vasireddy, R.J. Wallace Jr.)

DOI: <http://dx.doi.org/10.3201/eid2103.140577>

To the Editor: *Mycobacterium abscessus* is a ubiquitous, rapidly growing mycobacteria (RGM) found in water supplies, soil, and dust. *M. abscessus* is considered the most pathogenic and difficult to treat of the RGM and is most often associated with pulmonary, skin, and soft tissue infections; it has also been reported to cause ocular infections, otitis, lymphadenitis, arthritis, osteomyelitis, disseminated disease, and prosthetic valve endocarditis (1,2). Most prosthetic valve endocarditis cases have been fatal.

M. abscessus subsp. *massiliense* is 1 of 3 subspecies of *M. abscessus*. *M. abscessus* subsp. *massiliense* has an identical 16S rRNA gene sequence to the other 2 subspecies, *Mycobacterium abscessus* subsp. *bolletii* and *Mycobacterium abscessus* subsp. *abscessus*, but can be differentiated by *rpoB* and *erm41* gene sequencing (3,4). *M. abscessus* subsp. *massiliense* grows readily in blood culture media and on sheep's blood agar within 2–4 days. Care should be taken in interpreting Gram staining of isolates because RGM is not identifiable by this method and could be mistaken for corynebacteria or diphtheroids (5,6). Such isolates could be further tested by acid-fast staining and, if positive, sent to a reference laboratory for definitive identification and susceptibility testing.

Five cases of *M. abscessus* native valve endocarditis have been reported; 4 were fatal and 1 was lost to follow-up (1,5–9). One of the 4 fatal cases also involved the tricuspid valve and was associated with intravenous heroin abuse

(9). We report a case of *M. abscessus* subsp. *massiliense* native tricuspid valve endocarditis successfully treated with antimicrobial therapy and surgical debridement.

A 52-year-old man who used intravenous drugs was admitted to our hospital describing a 25-pound weight loss, fever, and night sweats. He reported injecting crushed opioid tablets mixed with tap water. He had tachycardia and pitting edema of the legs and feet. Laboratory data revealed elevated aminotransferase levels, thrombocytopenia, and opiates in the urine. Computerized chest tomographic scan showed cavitory right upper lobe and lingular nodules. Routine blood cultures (BacT/ALERT3D; bioMérieux, Marcy l'Etoile, France) of samples drawn at admission and on hospital day 3, before the initiation of antimicrobial drug therapy, grew acid-fast bacilli (AFB) in broth medium on days 3 and 4 of incubation. A transthoracic echocardiogram on hospital day 5 revealed a 1-cm vegetation on the tricuspid valve. An empiric regimen for RGM consisting of intravenous cefoxitin and amikacin and oral clarithromycin and moxifloxacin were administered. Based on preliminary (3-day) susceptibility test results showing susceptibility to amikacin, resistance to the quinolones, and intermediate susceptibility to cefoxitin, linezolid, and imipenem, the regimen was changed to tigecycline, linezolid, clarithromycin, and amikacin (10). Routine blood cultures on hospital days 11 and 17 were negative.

On hospital day 19, linezolid was stopped, and imipenem was added. A transthoracic echocardiogram on hospital day 31 showed the vegetation had enlarged to 1.5×0.5 cm. We concluded that antibiotics alone were unlikely to be curative; cardiac catheterization was performed on hospital day 38. On the basis of hemodynamic findings, the cardiologist inferred that valve replacement would be of no value and recommended valvectomy alone.

Surgery on hospital day 41 revealed a 2-cm nodule on each anterior and posterior leaflet and a 2–3 mm nodule on the septal leaflet of the tricuspid valve. The anterior and posterior leaflets were removed, and the septal leaflet was segmentally resected. Routine cultures of valve tissues, in which *M. abscessus* would have grown, were negative. Pathologic examination confirmed suppurative vegetations with numerous bacterial colonies consistent with AFB; AFB staining disclosed numerous mycobacteria (Figure, <http://wwwnc.cdc.gov/EID/article/21/3/14-0577-F1.htm>).

Identification and final susceptibilities of the RGM from the original blood culture isolate revealed *M. abscessus* subsp. *massiliense* by *hsp65* PCR and *erm* gene sequencing (4) and 14-day susceptibility to clarithromycin (10). *M. abscessus* subsp. *massiliense* has a nonfunctional (truncated) macrolide-inactivating gene (*ermA1*), and untreated isolates are susceptible to the macrolides (4).

Repeat chest tomographic scan on hospital day 69 showed nearly complete resolution of the RUL cavitory and lingular nodules/infiltrates. Tigecycline, amikacin,

imipenem, and clarithromycin were continued until hospital day 77, when amikacin was stopped because of moderate hearing loss. The patient was discharged without antibiotics after 2 months of postoperative antibiotic therapy. At follow-up visits 2 and 8 weeks later he was doing well except for peripheral edema. AFB and routine blood cultures drawn at both visits were negative. He is periodically seen in the cardiology clinic; his edema has resolved with diuretic therapy.

Cure of *M. abscessus* native valve endocarditis has not been previously reported. A case of *M. chelonae* native tricuspid valve endocarditis associated with a pacemaker lead was successfully treated with wire removal, valve debridement, and antimicrobial therapy (11). The patient in the current study likely acquired his infection from the tap water diluent he injected. Clinicians should consider the possibility of mycobacterial endocarditis when evaluating a septic patient with intravenous drug use history or cardiac prosthetic devices.

We successfully treated mycobacterial native tricuspid valve endocarditis with combination antimicrobial therapy and surgical debridement. The location of the infection in the tricuspid valve and favorable hemodynamics enabled debridement without implantation and the subsequent possibility of intraoperative infection of a prosthetic valve.

This study was supported in part by a grant from the Amon G. Carter Foundation.

References

- Griffith DE, Aksamit T, Brown-Elliott BA, Catanzaro A, Daley C, Gordin F, et al. An official ATS/IDSA statement: diagnosis, treatment, and prevention of nontuberculous mycobacterial diseases. *Am J Respir Crit Care Med.* 2007;175:367–416. <http://dx.doi.org/10.1164/rccm.200604-571ST>
- Liebeskind DS, Ostrzega N, Wasterlain CG, Buttner EA. Neurologic manifestations of disseminated infection with *Mycobacterium abscessus*. *Neurology.* 2001;56:810–3. <http://dx.doi.org/10.1212/WNL.56.6.810>
- Adékambi T, Colson P, Drancourt M. rpo B-based identification of nonpigmented and late-pigmented rapidly growing mycobacteria. *J Clin Microbiol.* 2003;41:5699–708. <http://dx.doi.org/10.1128/JCM.41.12.5699-5708.2003>
- Kim HY, Kim BJ, Kook Y, Yun YJ, Shin J H, Kim BJ, et al. *Mycobacterium massiliense* is differentiated from *Mycobacterium abscessus* and *Mycobacterium bolletii* by erythromycin ribosome methyltransferase gene (*erm*) and clarithromycin susceptibility patterns. *Microbiol Immunol.* 2010;54:347–53. <http://dx.doi.org/10.1111/j.1348-0421.2010.00221.x>
- Williamson JC, Miano T, Morgan M, Palavecino E. Fatal *Mycobacterium abscessus* endocarditis misidentified as *Corynebacterium* spp. *Scand J Infect Dis.* 2010;42:222–4. <http://dx.doi.org/10.3109/00365540903384158>
- Larkin JA, Shashy RG, Gonzalez CA. Difficulties in differentiating a rapidly growing *Mycobacterium* species from diphtheroids in an immunocompromised patient. *ClinMicrobiol Newsl* 1997; 19:108–11. [http://dx.doi.org/10.1016/S0196-4399\(97\)82722-5](http://dx.doi.org/10.1016/S0196-4399(97)82722-5)

7. Al-Benwan K, Ahmad S, Mokaddas E, Johny M, Kapoor M. Diagnosis of endocarditis caused by *Mycobacterium abscessus*. *Ann Saudi Med*. 2010;30:408–11.
8. Corrales-Medina V, Concha R, Simkins J, Sanchez M, Baracco G. Native valve endocarditis caused by rapidly growing mycobacteria: case report and review of the literature. *Scand J Infect Dis*. 2007;39:639–41. <http://dx.doi.org/10.1080/00365540601169745>
9. Tsai WC, Hsieh HC, Su HM, Lu PL, Lin TH, Sheu SH, et al. *Mycobacterium abscessus* endocarditis: a case report and literature review. *Kaohsiung J Med Sci*. 2008;24:481–6. [http://dx.doi.org/10.1016/S1607-551X\(09\)70005-1](http://dx.doi.org/10.1016/S1607-551X(09)70005-1)
10. Clinical and Laboratory Standards Institute. 2011. Susceptibility testing of mycobacteria, nocardia, and other aerobic actinomycetes; Approved Standard-Second Edition. CLSI document M24–A2. Wayne, PA: Clinical and Laboratory Standards Institute. Vol. 31, Number 5.
11. Galil K, Thurer R, Glatter K, Barlam T. Disseminated *Mycobacterium chelonae* infection resulting in endocarditis. *Clin Infect Dis*. 1996;23:1322–3. <http://dx.doi.org/10.1093/clinids/23.6.1322>

Address for correspondence: R. Gordon Huth, University of Texas Southwestern Residency Programs, 601 E 15th St, Austin, TX 78701, USA; email: ghuth@seton.org

***Rickettsia rickettsii* in *Amblyomma patinoi* Ticks, Colombia**

Álvaro A. Faccini-Martínez, Francisco B. Costa, Tatiana E. Hayama-Ueno, Alejandro Ramírez-Hernández, Jesús A. Cortés-Vecino, Marcelo B. Labruna, Marilyn Hidalgo

Author affiliations: Pontificia Universidad Javeriana, Bogotá, Colombia (Á.A. Faccini-Martínez, M. Hidalgo); Universidade de São Paulo, São Paulo, Brazil (F.B. Costa, T.E. Hayama-Ueno, M.B. Labruna); Universidad Nacional de Colombia, Bogotá (A. Ramírez-Hernández, J.A. Cortés-Vecino)

DOI: <http://dx.doi.org/10.3201/eid2103.140721>

To the Editor: *Rickettsia rickettsii* is the etiologic agent of Rocky Mountain spotted fever (RMSF), a highly lethal tick-borne rickettsiosis restricted to the Western Hemisphere (1,2). In Colombia, *R. rickettsii* was first reported during the 1930s, when 62 (95%) of 65 affected persons died of RMSF in Tobia town (Cundinamarca Department) (3), from where highly virulent strains of *R. rickettsii* were isolated through the inoculation of patient blood or of *Amblyomma cajennense* sensu lato (s.l.) extracts into guinea pigs (4). Thereafter, RMSF remained unnoticed in Colombia until the 21st century, when new outbreaks with high case-fatality rates were reported in different regions, including Villeta, a nearby locality of Tobia (1).

Recent studies have shown that *A. cajennense* s.l., widely distributed from the southern United States to Argentina, is actually a complex of 6 different species: *A. cajennense* sensu stricto (Amazonian region), *A. mixtum* (from Texas, USA, to western Ecuador), *A. sculptum* (northern Argentina, Bolivia, Paraguay, Brazil), *A. interandinum* (inter-Andean valley of Peru), *A. tonelliae* (dry areas of northern Argentina, Bolivia, and Paraguay), and *A. patinoi* (eastern cordillera of Colombia) (5). With this new classification, *A. patinoi*, originally described from Villeta, is the only species of this complex known to occur in the RMSF-endemic area of Cundinamarca, Colombia (5).

In August 2013, we collected 15 *A. patinoi* adult ticks from cattle in Naranjal village (5°3'31.52"N, 74°26'50.24"W), Villeta town, an area of Cundinamarca, Colombia, to which RMSF is endemic. Ticks were taken alive to the laboratory, where they were frozen at –80°C for further analysis. The 15 ticks were defrosted, surface sterilized with iodine alcohol, and processed individually by the shell vial technique for isolation of rickettsiae in Vero cells, as described (6). Infected cells were always incubated at 28°C. Rickettsiae were observed by Gimenez staining within cells (online Technical Appendix Figure 1, <http://wwwnc.cdc.gov/EID/article/21/3/14-0721-Techapp1.pdf>) from only 1 (inoculated from a female tick) of the 15 inoculated shell vials. This isolate was subjected to at least 7 Vero cell passages, each achieving >90% infected cells.

DNA was extracted from an aliquot of first passage–infected cells and tested by a battery of PCR protocols targeting fragments of the rickettsial genes *gltA*, *ompA*, and *ompB* and the intergenic regions RR0155-*rpmB*, RR1240-*tlc5b*, and *cspA-ksgA* (Table). We sequenced 1,106 bp, 512 bp, and 799 bp of the *gltA*, *ompA*, and *ompB* genes, respectively. By BLAST analyses (<http://www.ncbi.nlm.nih.gov/blast>), these sequences were 100% identical to corresponding sequences of *R. rickettsii* from Colombia and Brazil (GenBank accession nos. CP003306, CP003305). Generated sequences for 2 intergenic regions, RR0155-*rpmB* (228 bp) and RR1240-*tlc5b* (306 bp), were 100% identical to corresponding sequences of the same 2 *R. rickettsii* isolates from Colombia and Brazil. A 337-bp sequence of the *cspA-ksgA* intergenic region was 100% (337/337 nt) identical to *R. rickettsii* from Brazil (CP003305) and 99.7% (336/337) to *R. rickettsii* from Colombia (CP003306). Partial sequences from *R. rickettsii* generated in this study were deposited into GenBank and assigned nucleotide accession nos. KJ735644–KJ735649.

Whole-body remnants of the 15 ticks used to inoculate shell vials were also subjected to DNA extraction and processed by PCR for the rickettsial *gltA* gene (Table); only 1 tick (the one that provided the rickettsial isolate) contained rickettsial DNA, indicating a 6.6% (1/15) infection rate. We confirmed the taxonomic identification of this